Reply to: Holistic anaesthesia machine: Need for a task force

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DOI: https://doi.org/10.1097/EJA.0000000000000001

EJA

Eur J Anaesthesiol 2014; **31:**172–180

CORRESPONDENCE

Endotracheal tube cuff inflation with and without a pressure gauge to minimise sevoflurane pollution during intermittent positive pressure ventilation

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Published online 27 November 2013

Editor,

Possible health hazards from exposure to trace concentrations of inhalational anaesthetics cannot yet be definitively excluded.[1] We have previously reported that the concentrations of the commonly used volatile anaesthetic sevoflurane were significantly higher in the proximity of the intubated patient's mouth than in the breathing zone of the surgeon or the far-end corner of the theatre.[2] This observation contradicts the core function of the cuff, which is to seal the upper airway. Although previous in-vitro studies highlighted the possibility of fluid microleakage and its dependence on intra-cuff pressure,[3,4] less is known about whether proper inflation influences the per oral escape of sevoflurane. Therefore, the main objective of the present study was to answer the question whether the concentration of sevoflurane at the patient's mouth differed depending on whether the balloon was inflated under controlled conditions to 25 to 30 cmH_2O using a pressure gauge or empirically, that is under manual control of the pilot balloon only.

With institutional ethics approval (DEOEC RKET/IKET 2483–2006), we included 30 patients undergoing craniotomy after having given informed consent. In the first 15 patients, tracheal cuffs were inflated under control of the Rüsch Endotest system setting the intracuff pressure between 25 and 30 cmH_2O. In the second 15 patients, tracheal cuffs were inflated empirically, that is while estimating intracuff pressure based on the hardness of the pilot balloon. Cuff pressures were measured by an independent person. All cases used RüschFlex tracheal tubes with a low-pressure cuff made of polyvinyl chloride (PVC; Teleflex Medical GmbH, Kernen, Germany) with sizes of 8.5 and 7.5 for men and women, respectively. General anaesthesia was performed as previously outlined in detail.[2] During anaesthesia, we monitored peak pressure, plateau pressure, minute volume and end-tidal concentration of sevoflurane in order to explore possible relations to the degree of anaesthetic escape and thereby obtain hints to the origin of the release.

For the detection of airborne sevoflurane, we used a detection set-up that consisted of a portable air sampling pump (224–51TX Air Sampling Pump; SKC, Dorset, England), an integrated tube system and an absorber ampoule coupled to the tube system. The distal part of the tube was placed in the close proximity of the patient's mouth. Cumulative sampling of sevoflurane at the patient's mouth was restricted to the period starting from reaching equilibrium (i.e. when fractions of inhaled and exhaled are only marginally different) to the beginning of skin closure. After termination of sample collection, the ampoule containing the absorber underwent chromatographic assessment by an independent chemist (CP), as described previously.[2] Median sevoflurane concentrations and balloon pressure were compared using Mann–Whitney *U*-test and Student's unpaired *t*-test, respectively. Differences were considered statistically significant if $P < 0.05$. Calculations were carried out using Statistica for Windows software (StatSoft, Tulsa, Oklahoma, USA).

Under controlled conditions, intracuff pressures were successfully inflated to a pressure between 25 and 30 cmH_2O; mean [SD], 27.7 [1.9] cmH_2O. In contrast, when cuffs were inflated under manual control, intra-cuff pressures were found to be higher; 53.0 [17.0] cmH_2O ($P < 0.001$). Median (IQR) concentration of sevoflurane after inflating the endotracheal cuffs under manometer control was 1.0 ± 2.6 ppm, whereas under manual control was 0.79 ± 1.49 ppm ($P = 0.78$). Concentration of sevoflurane detectable at the patients' mouth showed no association with sevoflurane concentration at plateau pressure (Spearman's rank correlation coefficient −0.08, $P = 0.63$), but did show a significant association with the end-tidal concentration and the minute volume of sevoflurane (Spearman's rank correlation coefficient 0.53 to 0.55, $P = 0.003$).

The present study highlights that empirically inflated cuffs may result in intracuff pressures that frequently exceed the recommended limits of 25 to 30 cmH_2O. To avoid the unwanted impact of high cuff pressures on the tracheal tissue, cuff inflation should be performed under guidance of a manometer to ensure an ideal cuff pressure.[5]

Moreover, higher cuff pressures do not seem to promote a more complete sealing of the trachea or decrease environmental pollution and exposure of staff. Our observations are in line with previous in-vitro observations in bench-top models revealing fluid leakage across the endotracheal cuff even at intraballoon pressure of 60 cmH_2O.[4] This leakage can be ascribed to the presence of longitudinal folds within the cuff wall and this is particularly common in cuffs made of PVC. Major discrepancies between tracheal diameter and balloon size in the individual patient may increase the likelihood and number of longitudinal folds in the balloon wall.[6] In further exploring the source of sevoflurane causing the apparent local increase in its concentration at the patient's mouth, we also assessed associations with various key parameters of the actual ventilation. The only parameter that could be related to the sevoflurane concentration at the patient's mouth was the end-tidal sevoflurane concentration and the minute volume, suggesting that the locally increased concentration of the volatile anaesthetic may be linked to the ventilated lung.

With regard to the potential implications, it is worth pointing out that sevoflurane concentration at the patient's mouth sometimes exceeds the clinically significant threshold value of 2 ppm, suggesting that more investigations into this topic may be warranted.

Acknowledgements relating to this article

Assistance with the study: none.

Financial support and sponsorship: none.

Conflicts of interest: none.

Presentation: none.

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DOI:10.1097/EJA.0000000000000018

The right supraclavicular ultrasound view for real-time ultrasound-guided definite placement of a central venous catheter with a microconvex transducer

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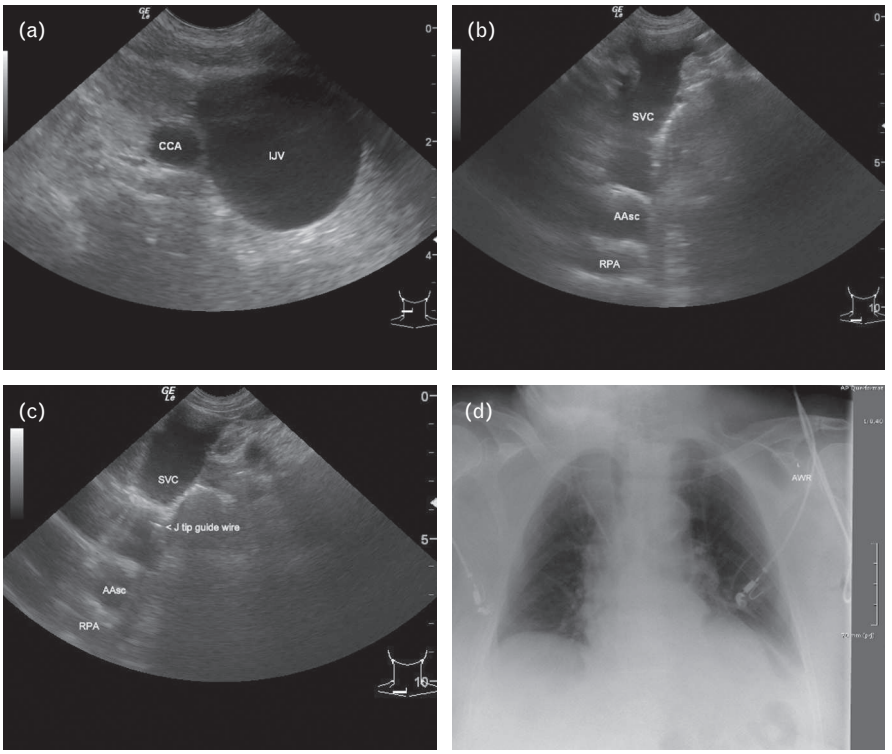
Published online 18 January 2014

Editor,

Ultrasound guidance for central venous catheter (CVC) access improves patient safety.[1] Most studies focus on the ultrasound-guided needle during venous puncture employing a short axis view of the internal jugular vein (IJV).[2–4] Further approaches to eliminate the need for a confirmatory chest radiograph include transthoracic views to visualise the CVC after placement.[2] However, definite positioning of the CVC in the superior vena cava (SVC) via a right supraclavicular view has not been evaluated up to date. The right supraclavicular view was described to visualise the great intrathoracic vessels including SVC, pulmonary arteries and ascending aorta.[5] We have now successfully assessed the positioning of a CVC in the SVC via the right supraclavicular view using a microconvex probe (8C-RS 4 to 10 MHz; GE Healthcare, Munich, Germany). To our knowledge, this is a novel approach for CVC placement and allows real-time ultrasound-guided CVC placement in a definite position in the SVC.

A 87-year-old woman (body weight 120 kg, height 140 cm, BMI 61.2 $kg\,m^{-2}$) with a history of arterial hypertension was scheduled for removal of a proximal femur nail. The patient provided written consent for the publication of this report. General anaesthesia was planned for the surgery including a CVC placement for postoperative intravenous antibiotic treatment. Due to unexpected pre-operative hypokalaemia (2.67 $mmol\,l^{-1}$), it was necessary to prevent potential arrhythmias being provoked by the CVC guide wire and the CVC itself. Thus, we employed a microconvex probe for ultrasound-guided puncture of the right IJV in a short axis view of the carotid triangle and visualisation of the SVC via the right supraclavicular view following induction of general anaesthesia, intubation and mechanical ventilation (Fig. 1a–b). The guide wire was introduced into the right IJV and the J-formed guide wire tip was identified in the right supraclavicular view in the SVC. Under direct ultrasound guidance, the tip of the guide wire was advanced into its final position in the distal part of the SVC using the ascending aorta and right pulmonary artery (RPA) as landmarks (Fig. 1c). Anatomically, the RPA crosses behind the SVC at the cavo-atrial junction. The CVC was inserted using the Seldinger technique

Fig. 1



(a) Transverse view of common carotid artery (CCA) and internal jugular vein (IJV) with a microconvex probe in the right carotid triangle. (b) Right supraclavicular view: superior vena cava (SVC), ascending aorta (AAsc), right pulmonary artery (RPA), scale in cm. Note body mark for position of the ultrasound probe. (c) SVC with J tip of the guide wire. (d) Postoperative chest radiograph with indwelling central venous catheter (CVC) in SVC.

until it reached the tip of the guide wire by reading the guide wire marks. The CVC had a final insertion depth of 14 cm. At this point, the guide wire was removed and the catheter was sutured at skin level. The patient did not experience cardiac arrhythmias at any time during the procedure. Postoperative chest radiograph showed a correct placement of the CVC in the SVC (Fig. 1d). We conclude that real-time definite central positioning of a CVC can be successfully carried out via the right supraclavicular view using a suitable ultrasound probe such as a microconvex or sector probe that allows sufficient tissue penetration. This novel approach can be conducted by a single operator in real-time using one single transducer for venous puncture and CVC positioning.

This concept should be further evaluated in a prospective observational study comparing ultrasound-guided definite CVC positioning with chest radiograph, intracardiac ECG and transesophageal echocardiography for control of CVC position, respectively. Study operators need to be trained in proper identification of vascular structures via the supraclavicular view. This study should also explore potential limitations of this concept including visibility of the IJV and sterility considerations.

Acknowledgements relating to this article

Assistance with the study: none.

Financial support and sponsorship: none.

Conflicts of interest: none.

Presentation: none.

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DOI:10.1097/EJA.0b013e3283642ae7

Transient paraplegia after epidural catheter removal during low molecular heparin prophylaxis

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Published online 7 January 2014

Editor,

We would like to briefly report on a case of transient paraplegia that occurred soon after the removal of a thoracic epidural catheter and resolved spontaneously. The patient provided informed consent for the publication of this report.

A 67-year-old woman, weight 43 kg, underwent combined epidural (T10 to T11 catheterisation) and general anaesthesia for upper oesophageal cancer removal. After admission to the ICU, due to some bleeding from the wound, thromboprophylaxis was not administered on the day of surgery. She received only epoprostenol sodium 5 ng kg^{-1} min^{-1} intravenously (i.v.) in order to improve the microcirculation at the colonic anastomotic site. Neither surgical complications nor signs of liver dysfunction were detected while in ICU, only mild renal impairment that occurred in the days following. On the third postoperative day, epoprostenol was withdrawn and the patient was transferred to the ward under low molecular weight heparin (LMWH) prophylaxis (dalteparin 3000 IU per day). That night the subcutaneous dalteparin was increased to 5000 IU per day due to immobility, and from the fourth day, ketorolac 30 mg three times daily was also given. In the absence of abnormalities of both platelet count and routine coagulation tests, on the fifth day, 14 h after the last dalteparin 5000 IU administration, the epidural catheter was removed without consulting an anaesthesiologist. At the time of epidural catheter removal, the serum creatinine level was 59.2 mol l^{-1}, and the calculated glomerular filtration rate (according to the Cockcroft-Gault formula) was more than 55 ml min^{-1}. The last dose of ketorolac was administered approximately 6 h earlier.

A few minutes after catheter removal, the patient complained of severe, persistent pleuritic-like chest pain, barely responsive to tramadol. Less than 1 h later, she perceived numbness and loss of muscle strength of both lower extremities, rapidly followed by flaccid paraplegia. Neurological examination showed a total loss of mobility in the left and right lower limbs and an almost complete sensory deficit reaching to the T10 dermatome (Frankel Grade A status). MRI, promptly performed, revealed a large posterior epidural blood collection extending from T7 to T12, causing displacement and compression of the spinal cord.

Within a few minutes following the MRI, while waiting for consultation with the spinal surgeon, she began to recover, at first from the sensory deficit of the lower limbs and then gradually also from the motor deficit. At 2 h, the return of normal movement and sensation was complete (Frankel Grade E status). Careful neurological examination was carried out later, but no recurrence of sensory or motor deficit was observed. On the sixth day after the event, in the complete absence of neurological impairment, MRI showed a reduction in thickness of the posterior epidural hematoma from T7 to T12, with a thin perimedullary liquid film. No compression of the spinal cord was detected. The patient was then discharged home with no sequelae.

Thoracic epidural catheterisation is an accepted perioperative analgesic technique in patients undergoing oesophagectomy.[1] The higher dose of LMWH required in individuals at a high risk of thrombosis has raised concerns regarding the increased risk of neuraxial bleeding. Epidural haematoma formation following catheter removal in patients receiving LMWH is a well known complication but seems to be exceptional when it occurs after a minimum time interval of 10 to 12 h following administration.[2–5] However, renal dysfunction influences LMWH elimination, and a bioaccumulation of LMWH due to reduced creatinine clearance in elderly patients with low lean body mass might affect spontaneous haemostasis.[6] In our patient, the association of 5000 IU per day dalteparin and ketorolac before catheter withdrawal greatly augmented the risk of spinal bleeding. It has been suggested that LMWH may have a dose-dependent bleeding potential,[7] but a 5000 IU per day dose of dalteparin is usually recommended in high-risk patients undergoing major abdominal surgery, irrespective of weight.[8]

Although the terminal plasma half-life of ketorolac is approximately in the range of 4 to 6 h, its clearance in this patient with mild renal insufficiency was most likely reduced. A significant interaction between ketorolac and dalteparin to lengthen the bleeding times has been reported.[2,9] In accordance with the recent Nordic guidelines for neuraxial blocks in disturbed haemostasis,[2] the recommended interval between the last dose of ketorolac and epidural catheter removal should be approximately 24 h (Recommendation grade D; evidence category IV).

Unfortunately, the resident who removed the catheter was unaware of the potentiation of anticoagulation induced by NSAIDs in patients receiving LMWH.

The onset of the neurological deficit occurred very quickly, presumably because the initial bleeding in the epidural space was considerable enough to induce a very rapid spinal cord compression. Rapid resolution occurred

probably because the bleeding was short-lived, allowing the spread of the initial collection of blood to relieve the compression. It can also be hypothesised that, due to an open or partially open spinal canal at the site of epidural catheterisation (thoracic level), some blood may have moved out of the epidural space through the intervertebral foramina, thus resolving the compression.

Although early paraplegia has often been reported, the speed of complete spontaneous recovery from both sensory and motor deficit appears quite unusual.[10,11] Without this, immediate surgery may have been needed.

Given the seriousness of spinal cord injury, care should be employed in performing regional anaesthesia in renal impairment with LMWH prophylaxis. Vigilant clinical judgement and sensible 'real time' coagulation tests are required before removing an epidural catheter in the presence of renal dysfunction and drugs known to increase the risk of bleeding.

Acknowledgements relating to this article

Assistance with the letter: none.

Financial support and sponsorship: none.

Conflicts of interest: none.

Presentation: none.

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DOI:10.1097/EJA.0b013e328364567b

Breaking the needle

A rare complication on EZ-IO removal

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Published online 18 January 2014

Editor,

The intraosseous access is a rather old approach to the vascular system, but has gained renewed attention during the last few years and is now considered standard in special circumstances.[1,2] Recent innovative technical solutions facilitate puncture as compared to the classic Cook or Jamshidi needles. The currently applicable resuscitation guidelines state that if intravenous access cannot be achieved, drugs should be given via the intraosseous route.[1] The EZ-IO system (Vidacare Corporation, San Antonio, TX) was first described in medical literature in 2004 and has gained wide propagation due to its ease of use and safety.[3,4] A recent meta-analysis identified extravasation, osteomyelitis, compartment syndrome and fractures as possible, but rare, complications.[5]

We report a previously unencountered complication with the removal of the EZ-IO intraosseous needle that should be able to be avoided by strict abidance by the manufacturer's directions for use. It is recommended to connect a Luer-lock syringe to the positioned intraosseous needle and to apply more rotation than pull onto the catheter on removal.

In a trauma course demonstration setting, a 38-year-old male volunteer (one of the authors) agreed to a training usage of an EZ-IO intraosseous needle. After effortless and uncomplicated placement on site of the proximal tibia and test-infusion of a small amount of isotonic saline, the device was to be removed. Divergent from the manufacture's suggestions, the still sterile steel stylet was put back into place to achieve better grip for a manual pull-out. Under steady pull in strict axial alignment and gentle clockwise turn, the needle broke away from the plastic connector, followed by passive blood emersion from the still locked needle. Bleeding could be stopped by intermediate coverage with sterile gauze. The needle was then carefully and successfully extracted with a pair of combination pliers and a notable amount of strict axial force. The volunteer's tibia is in good shape and shows no signs of further damage.

For catheter removal, the company (Vidacare) recommends connecting a Luer-lock syringe and to rotate it clockwise while using traction.[6] It is emphasised that the catheter should not be rocked or bent during removal. A careful analysis of the video recording of the removal manoeuvre revealed no major rocking or bending. More rotation could have been applied instead of intense pull, although a visible flick of the remover's wrist indicates at least some gyration. The reinsertion of the stylet to ensure grip might have added rigidity to the catheter needle, though we presume that Vidacare advises the use of a syringe, because the stylet will usually have already been disposed of.

The access to the toolbox of a fire department alleviated the solution of our problem, but might be inappropriate in other settings.

Acknowledgements relating to this article

Assistance with the letter: none.

Financial support and sponsorship: none.

Conflicts of interest: none.

Presentation: none.

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DOI:10.1097/EJA.0b013e328364fe51

Analgesic efficacy of ultrasound-guided adductor canal blockade after arthroscopic anterior cruciate ligament reconstruction

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Published online 12 October 2013

Editor,

We read with interest the article by Espelund *et al.*[1] entitled 'Analgesic efficacy of ultrasound-guided adductor canal blockade after arthroscopic anterior cruciate ligament reconstruction: A randomised controlled trial'. The authors have compared the use of adductor canal block and multimodal analgesia with multimodal analgesia alone for analgesia following anterior cruciate ligament repair with ipsilateral hamstring graft. The methodology is exemplary and the results are novel in showing very low mean pain scores at rest, movement and standing at 2 h. The study deviates from others in demonstrating very low pain scores (median VAS of 20 mm on standing at 2 h) with similar levels of dispersion in both the study groups. Although adductor canal block has been sparsely evaluated for this surgery, previous studies evaluating femoral nerve blocks for anterior cruciate ligament repair have reported higher pain scores than that noted in the present study and a clear benefit from blocks.[2,3] Dahl *et al.*[4] reported similar pain scores as the current study without employing blocks wherein they evaluated COX-2 inhibitors with or without steroids, combined with a cryo-cuff unlike, the present study. Although not all patients require blocks for analgesia, factors predicting their need are still a topic of research and have even compelled some researchers to develop models based on machine-learning to predict who would benefit from them.[5]

The observed results, although difficult to explain as elaborated by the authors, the inadequate sample size, the method of block performance and the presence of a confounding variable need to be investigated further. First, the degree of reduction in pain that is clinically meaningful needs to be decided before choosing the sample size in order to determine the significance of nerve blocks. The authors assumed a 50% reduction in pain scores in the experimental group to be meaningful, which obviously resulted in a low sample size (22 patients per group), a skewed distribution and an inability to show a significant difference between the two groups. If the authors were to calculate the sample size to achieve a 25 or 30% improvement in pain scores, they would need 99 or 44 patients per group, respectively. What is really surprising is the very low pain scores noted in either group. Such pain scores are desirable and make performance of any regional technique unnecessary. Another possibility for a lack of difference between the groups may be the method of performing the block. Also, the success rate of the block was not tested, which is unusual in clinical practice. The authors of the study performed the blocks in the postoperative period; this may not be possible in the clinical practice due to the presence of braces and surgical dressings which make appreciation of anatomical structures difficult.

Bushnell *et al.*[6] did not comment on the site of pain; the graft donor site can be a significant source of pain even with an effective femoral nerve block. Hence, in the presence of a confounding factor such as pain in the nonblocked area (graft donor site), comparing and

commenting on pain scores could be misleading. If the patients requested and received enough analgesics to achieve pain control at the donor site where the adductor canal block will have no effect, there will be no difference in pain scores or analgesic consumptions. Attractive strategies to cover the donor site such as a block of the anterior division of obturator nerve or intramuscular injections into the donor muscles near the motor end-plate zones similar to those performed for botulinum toxin injection[7] need to be evaluated. Cumulative rescue analgesic frequency over time would have provided a better idea about the effect of block on analgesic consumption.

Acknowledgements relating to this article

Assistance with the letter: none.

Financial support and sponsorship: none.

Conflicts of interest: none.

Presentation: none.

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DOI:10.1097/01.EJA.0000434969.37753.13

Reply to: analgesic efficacy of ultrasound-guided adductor canal blockade after arthroscopic anterior cruciate ligament reconstruction

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Published online 18 January 2014

Editor,

We thank Drs Sondekoppam and Ganapathy for their interest in our trial and their careful reading thereof.[1] As described in our article,[2] we were also surprised about the results revealing very low postoperative pain scores during the first 24 h after surgery.

We considered a 50% reduction in pain scores in the intervention group to be clinically relevant. This threshold was decided before estimating the sample size and was based on our clinical experience, previous studies and an assumption of a much higher pain score than the one observed. On the basis of our results, we cannot conclude that there is no effect of the adductor canal blockade; we just have to accept that the patients in this trial receiving a basic analgesic recipe of paracetamol and ibuprofen in both groups did not have sufficient pain to detect an effect of the blockade.

We are aware of the problem about the success rate of the block not being tested. In order to avoid un-blinding of the study, no test of sensibility of the lower extremity (the normal procedure before discharge of patients) was made. We decided to let the risk of introducing a potential confounding factor prevail the wish for knowing the success rate of the performed blocks. We do recommend testing the effect of the adductor canal blockade in routine care.

As we interpret our data, the major challenge in this trial was the fact that all patients responded with unexpectedly low pain scores.

We expected this specific surgical intervention to cause pain at the hamstring donor site, as well as intra-articular/periarticular pain. We asked the patients to evaluate their 'overall' pain level (pain related to the surgical procedure); it would be of no benefit to relieve, for example, their intraarticular pain only if the patients were still in pain due to surgical trauma at the hamstring site. However, ketobemidone consumption in both groups of patients was modest (7.5 vs. 5.0 mg in the control vs. the ropivacaine group, respectively) during the first 24 h after surgery indicating low pain levels in general.

Our clinical experience regarding postoperative pain after anterior cruciate ligament reconstruction prior to this trial was based on data from different surgeons, and data from the literature. One surgeon performed the vast majority of the surgical procedures in our trial. We therefore must assume that the surgical intervention performed in the two groups of patients was comparable. But we also have to consider whether the surgical technique could have an impact on the degree of postoperative pain. Maybe it is of importance where and how the tendon from the sartorius or gracilis muscle is being harvested; for example whether or not the periosteum is affected when harvesting the donor

tendon. Evaluating the outcome of this trial makes us consider this as a possible important factor for the observed results.

Acknowledgements relating to this article

Assistance with the letter: none.

Financial support and sponsorship: none.

Conflicts of interest: none.

Presentation: none.

References

1 Sondekoppam RV, Ganapathy S. Analgesic efficacy of ultrasound-guided adductor canal blockade after arthroscopic anterior cruciate ligament reconstruction. *Eur J Anaesthesiol* 2014; **31**:172–180.

2 Espelund M, Fomsgaard JS, Haraszuk J, *et al.* Analgesic efficacy of ultrasound-guided adductor canal blockade after arthroscopic anterior cruciate ligament reconstruction: a randomised controlled trial. *Eur J Anaesthesiol* 2013; **30**:422–428.

DOI:10.1097/01.EJA.0000434968.60623.43

Holistic anaesthesia machine

Need for a task force

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Published online 20 November 2013

Editor,

I have read with great interest the recently published Editorial on a holistic anaesthesia machine by Professor Biro.[1] The anaesthesia system, described therein, has been my dream from the day of my very first general anaesthetic. However, I cannot share Professor Biro's faith in the industry to develop such a device, or to put it maybe a little bit better – to develop it correctly. If we want it to be a system that can integrate several drugs from different companies, and at the same time being able to function in dissimilar anaesthesia architectures, then there are many conflicts of interests to be taken into account. Therefore, it is unlikely that industry itself will produce such a device.

And even if the industry does, there is always the risk of closed algorithms as it is the case with bispectral index (BIS) monitors, thereby limiting its usefulness and preventing scientists around the globe from improving them.

I am deeply convinced that we need a new holistic anaesthesia architecture and that it has to be developed by a Task Force created by an independent scientific society such as the European Society of Anaesthesiology. The Task Force should afterwards also coordinate and provide funding for further research that would eventually lead to full implementation of this architecture in a commercially available system. Of course, at this stage, close cooperation with our industry partners would be necessary.

The avenues that would open for us if we could implement such a system in our clinical practice seem huge. Therefore, I would, once again, plea for a Task Force of the European Society of Anaesthesiology on this account.

Acknowledgements relating to this article

Assistance with the letter: none.

Financial support and sponsorship: none.

Conflicts of interest: none.

Presentation: none.

Reference

1 Biro P. A plea for an independent holistic anaesthesia delivery system. *Eur J Anaesthesiol* 2013; **30**:97–101.

DOI:10.1097/EJA.0000000000000002

Reply to: holistic anaesthesia machine

Need for a task force

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Published online 14 November 2013

Editor,

I am delighted to know that there is at least another dreamer, who understands and supports the rationale and the necessity for a holistic and independent anaesthesia system.[1] And I am neither surprised nor offended that my fellow dreamer, Skitek, questions my faith in the industry to develop such a system. Furthermore, I have to admit that I have been too optimistic so far, maybe driven by the somewhat naive belief that a superior system or device might become a reality if there was an outlook for economic success with it.[2] Probably in this case, more than just a profitable business case is necessary to trigger the desired progress. My decade-long, unsuccessful search for an industrial partner in this respect gives credence to his argument. However, dreams and hopes have the inbuilt tendency to surprisingly materialise, even against all odds, after some time.

As suggested by Skitek the establishment of a task force in a supranational anaesthesiological body such as the European Society of Anaesthesiology (ESA) or the European Society for Computing and Technology in Anaesthesia and Intensive Care (ESCTAIC) would be a suitable approach. Let us hope that more voices for the creation of such a Task Force will be heard in the future, thus leading to new developments on the ground.

Acknowledgements relating to this article

Assistance with the letter: none.

Financial support and sponsorship: none.

Conflicts of interest: none.

Presentation: none.

References

1 Skitek K. Holistic anaesthesia machine: need for a task force. *Eur J Anaesthesiol* 2014; **31**:172–180.
2 Biro P. A plea for an independent holistic anaesthesia delivery system. *Eur J Anaesthesiol* 2013; **30**:97–101.

DOI:10.1097/EJA.0000000000000001